- Identify the common and classic symptoms of pulmonary embolism
- Describe the prehospital care for a patient experiencing pulmonary embolism

A 48-year-old male patient has phoned 9-1-1 for severe respiratory distress. You arrive to find an anx
take you to the second floor of a two-story flat. Hustling up the stairs, the neighbor advises you that h
Henry, his neighbor, after Henry had surgery to repair his right lower leg following a motorcycle accide
for five days.

You find Henry seated upright on the side of his bed in a tripod position, with pale and diaphoretic ski
minute. In one-word answers he denies any medical history other than his recent leg surgery, reports
taking morphine for pain from the surgery. He specifically denies any prior breathing problems.

Working quickly, your partner administers oxygen 15 lpm via nonrebreather mask while you determin
min. with a narrow-complex tachycardic rhythm on the cardiac monitor. His SpO_2 is 78% on room air,
74/50 mmHg. Henry is unable to walk, so your partner leaves to retrieve the stair-chair while you esta
right antecubital region and begin a 500-cc normal saline fluid bolus. By the time your partner returns
increased to 84% on oxygen, and you've determined that he has slight crackles in the bases of both l
cyanosis to the lips and is negative for chest pain, abdominal distress and peripheral edema. His leve
appears to be increasing.

Introduction

Acute pulmonary embolism (PE) is a life-threatening emergency during which an obstruction occurs i
arteriole. Pulmonary embolism occurs in more than 650,000 people annually.[1] While more than 75% o
stable, around 10% die within the first few hours of symptom onset, and overall mortality remains arou
patients with PE rapidly deteriorating in just a few hours, PE remains the second-leading cause of suc
sudden cardiac death).[3] Thus it is important for all prehospital professionals to understand when to su
acute pulmonary embolisms.

One out of every 1,000 Americans is diagnosed with an acute PE annually.[2] This rate rose each year
improved ED diagnostic capabilities, including the availability of CT scanners.[2,3] Most experts believe
attributed improved recognition of previously missed PEs, not a true rise in the overall occurrence.

Women 55 and older are at greater risk for PE than men of the same age, but death rates are 20%–3
also an interesting correlation between PE and race: The incidence and mortality from PE is nearly 50
whites, and the mortality is 50% higher for whites than it is for other races (Asians, South Americans,

Between 60%–80% of patients diagnosed with PE first have a deep vein thrombosis (DVT). A DVT is
deep vein and occurs in 10%–13% of all patients who have been on bed rest for seven or more days,
with pulmonary disease who are on bed rest for three or more days.[3] Thus most patients ordered exte
on DVT prophylaxis to help mitigate the risk of DVTs developing.

Examples of DVT prophylaxis include anticoagulation medications and the application of compressio
mmHg of pressure. When a DVT is diagnosed early and treated with anticoagulants, the patient has m
PE than they do from anticoagulant-associated fatal hemorrhage (less than 1%).[4] While an in-depth d

Atrial thrombus formation most frequently occurs when the heart is in atrial fibrillation. A thrombus is a
a vessel and remains at its place of origin, while an embolism occurs when a piece of that clot breaks
bloodstream and becomes lodged in another part of the body. In the case of a pulmonary embolism o
heart thrombus, the blood clot travels up the inferior vena cava, through the right side of the heart and
system (*Figure 2*). Blood clots are the most common emboli, but emboli can also occur from fatty tiss
released when a bone fractures, as well as from air, a piece of tumor, sheared IV catheter or any othe
the bloodstream.

Thrombosis, the formation of a blood clot, normally occurs to help tamponade hemorrhage following i
the vessels, thrombosis usually takes place on the valves in the lower extremities. The thrombus then
fibrin aggregate, leading to thrombus formation. When the thrombus occurs in a deep vein, it is consid
occlusive thrombus if it interrupts blood flow.

When a piece breaks loose, an embolism develops. Emboli that break free in the venous system trav
continuously gain diameter size until they reach the heart; thus it is unlikely for emboli to occlude a ve
through the heart. Once the embolism passes through the heart, it enters the pulmonary arteries, whe
decrease in size until they become the pulmonary capillary bed. The embolus then becomes lodged o
the artery through which it is traveling. Small emboli tend to travel distally within the pulmonary artery
occlusion, which can produce minimal symptoms that include pleuritic chest pain that develops from t
inflammatory response to the emboli. Large emboli may lodge at the bifurcation of the main pulmonar
lobar artery branches, resulting in rapid hemodynamic instability.

Largely impacted by the emboli's original size, the acute pulmonary embolism is characterized as eith
central PE is found in the central vasculature of the pulmonary tree, which includes the main pulmona
right and left interlobar arteries, left upper lobe trunk, right middle lobe artery, and right and left lower
pulmonary arteries are involved, or when the PE causes hemodynamic compromise, it is considered
pulmonary embolism (*Figure 3*) bridges across bifurcation of the main pulmonary artery and can inter
lungs, causing rapid deterioration and death. Peripheral PEs lodge in the segmental and subsegment
size by comparison, and because they disrupt little blood flow, they typically do not cause hemodynar
peripheral PEs occur in the lower lung lobes and are multiple in nature, meaning more than one embo

The physiologic effects of a PE are largely determined by the embolism's size and where it becomes
pulmonary artery, the PE disrupts distal blood flow, inhibiting the exchange of oxygen and carbon diox
backup within the cardiovascular system. As a result, clinicians are likely to see a manifestation of pri
cardiovascular symptoms.

Within the respiratory system, PE leads to:

- Increased dead air space (VQ mismatch);
- Hypoxemia;
- Hyperventilation;
- Pulmonary infarction.

Within the cardiovascular system, PE can lead to:

symptoms when a PE forms, and particularly when patients are suffering from a small embolism, thei
over time, with mild shortness of breath and intermittent mild chest pain. The most common are:

- Acute shortness of breath;
- Cough;
- Chest pain;
- Tachycardia;
- Tachypnea;
- Signs of DVT.

Complete a thorough set of vital signs and accurately count respirations; nearly all patients present w
tachypnea.[3] Determine a baseline SpO_2 and monitor for change with oxygen administration. Do not b
a suspected PE does not experience a significant rise in SpO_2 with supplemental oxygen. Patients m
tachycardia, so establish cardiac monitoring as a part of your assessment and perform a 12-lead EC
identify new-onset atrial fibrillation.

Pulmonary embolism won't cause ST-segment elevation or depression, but it is likely to cause chang
strain, which include non-sinus rhythms, incomplete or complete right bundle branch block, the S1Q3
inversions in leads V1–V2, V1–V3 or V1–V4. A multicenter trial that reviewed more than 6,000 12-lea
presence of these ECG changes, consistent for right heart strain, increased the odds a patient withou
disease was experiencing a pulmonary embolism.[6] The S1Q3T3 pattern is a large S-wave in lead I, a
inverted T-wave in lead III. While the absence of these changes doesn't rule out PE, their presence is
pulmonary embolisms are defined as presenting with a systolic blood pressure less than 90 mmHg; th
dying within hours and should be considered critically unstable. Patients with massive PE are in shoc
peripheral perfusion and present pale, diaphoretic and often with mental status changes.

Perform a thorough head-to-toe examination. Neurological symptoms are typically only found when p
PE. They may include seizures, syncope (most common) and mental status changes. On the neck, in
distention. Listen to lung sounds; many patients with PE develop mild pulmonary edema as a result o
artery pressures. In addition, they may experience a nonproductive cough, and lung sounds may reve
the bases. Hemoptysis can occur, although it is rare.

One clue to suspecting PE in shock patients is the presence of a systolic murmur louder on inspiratio
border. This suggests tricuspid valve regurgitation. Abdominal pain is more likely when the PE results
pelvic veins. When examining the extremities, pay close attention for evidence of a deep vein thromb
DVT is unlikely to lose pulses, but patients may have nonspecific leg pain and tenderness (particularl
increased warmth and swelling isolated to the affected extremity. Pay close attention to the patient's s
clammy skin are common, and peripheral cyanosis in the hands and feet may rapidly progress to cen
neck and trunk.

It's important to consider PE whenever a patient experiences shortness of breath or hypoxia. But bec
not a disease but rather a complication of other coagulopathy, illness or injury elsewhere in the body,
diagnoses that can more easily explain the symptoms before treating for PE. Many illnesses and dise

because it lacks sufficient sensitivity or specificity to do so.

In one study of 270 patients with an ED diagnosis of pulmonary embolism, an elevated serum lactate with high risk of death and adverse outcomes independently from shock, hypotension or elevated trop

An arterial blood gas can be used to determine how well any patient with a respiratory complaint is ox oxygen delivery. However, an ABG should rarely be used to rule in or out pulmonary embolism, as it i hypoxemia. One exception to this would be patients with no known pulmonary disease who develop s (especially following surgery); in these patients a low PO_2 has a high predictive value for pulmonary e

While not a laboratory value, end-tidal CO_2 ($EtCO_2$) is a popular prehospital assessment tool for patie complaints. And while it may seem reasonable to consider $EtCO_2$ in the assessment of patients susp analysis of 14 trials determined it has a probability of less than 10% of accurately indicating pulmonar If you use it, expect a patient with a suspected PE to have a normal (35–45 mmHg) to slightly low EtC tachypnea, and a normal waveform.

Treatment

Prehospital treatment of acute pulmonary embolism is driven by patient stability and focuses on early measures. Patients with small pulmonary embolisms generally only require comfort care. Manage pai protocols; avoid drugs such as morphine, which may exacerbate hypotension. Unstable patients with aggressive intervention to protect the ABCs.

Administer all suspected PE patients oxygen to maintain SpO_2 above 94%. This may be accomplishe some patients, while others will remain hypoxic on a nonrebreather mask. Patients with persistent hyp noninvasive positive-pressure ventilation (CPAP or BiPAP). While this will not correct the underlying p oxygen delivery to the blood that's reaching the lungs without harming the patient. The benefit of impr outweigh the risk of slight blood pressure decline in patients with borderline blood pressures. Patients level of consciousness need early advanced airway management, including rapid sequence intubatio performed, consider adding a PEEP valve onto the bag-valve mask and ventilate the patient with sup maximize oxygen delivery to blood reaching the lungs.

Early IV access is important, as patients with PE who are going to deteriorate are most likely to do so Establishing large-bore IV access allows for the rapid administration of IV fluids; treat hypotension wit fluids to help increase stroke volume and cardiac output. If the patient's blood pressure does not resp initiate a vasopressor. Norepinephrine is the preferred first-line vasopressor for PE patients, as dopam already-present tachycardia, reducing the ventricular filling time and leading to a further decrease in s

Transport teams with anticoagulants such as Lovenox and heparin should consider early administrati of pulmonary embolism when not contraindicated. The American College of Chest Physicians recomm anticoagulation for any suspected PE patient, as doing so reduces mortality. In these patients intraven is preferred, as it will not interfere with fibrinolytic therapy, should it be required.[9] Lovenox administrat have a properly functioning renal system, so in the prehospital setting it is best avoided in any patient disease.

assessment and ruling out other, more likely illnesses. Diagnosing a PE requires a high index of susp
determining a PE risk assessment score. When treating a patient suspected to have a PE, protect the
adequate oxygen and anticipate the potential for rapid patient deterioration. Be prepared to intervene

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